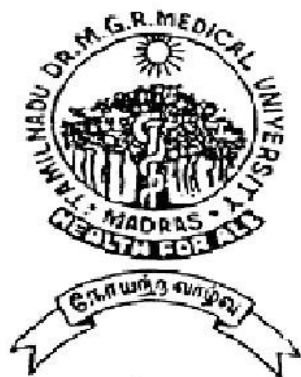


STUDY OF SEIZURES IN ELDERLY

DISSERTATION SUBMITTED FOR

BRANCH – I . D.M. (NEUROLOGY)

AUGUST 2008



THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled “**Study of seizures in Elderly**” is bonafide record work done by **Dr. D. CHEZHIAN** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for DM, Branch I – Neurology.

Dr.M.Chandrasekaran.

M.D.D.M.(Neuro)

Professor of Neurology,
Dept. of Neurology and Neuro Surgery,
Madurai Medical College,
Madurai

Dr.S.Manoharan.

M.Ch.,(Neuro)

Professor & Head of the Dept.
Neurology and Neuro Surgery,
Madurai Medical College
Madurai.

DECLARATION

I **Dr. D. CHEZHIAN** solemnly declare that the dissertation titled “**STUDY OF SEIZURES IN ELDERLY**” has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of D.M. degree Branch – I (Neurology) to be held in August 2008.

Place : Madurai

Dr. D. CHEZHIAN

Date :

ACKNOWLEDGEMENT

It gives me great pleasure to acknowledge all those who guided, encouraged and supported me in the successful completion of my dissertation.

First and foremost, I express my gratitude to Dr.S.M. Sivakumar. M.S. Dean (In-charge), Government Rajaji Hospital and Dr. M. Shanthi, M.D. Dean (In-charge), Madurai Medical College, Madurai for granting permission to utilize the hospital materials for conducting this study.

I express my deep sense of gratitude and sincere thanks to our respected and beloved Chief Dr. M. Chandrasekaran, M.D. D.M., Professor of Neurology, Madurai Medical College, Madurai for his teaching, constant motivation and guidance in the conduct of this study.

I am extremely thankful to Prof. Dr. S. Manoharan. M.ch., Head of the department of Neurology & Neurosurgery, Madurai Medical College, Madurai for his constant encouragement and support.

I express my sincere thanks to Prof. Dr. A. Ayyappan. M.D., Head of the Department of Medicine, Madurai Medical College, Madurai for allowing me to collect the case materials and for his guidance.

I express my sincere thanks and gratitude to our Assistant Professors Dr. T.R. Gnaneswaran. M.D.D.M., Dr.B. Sridharan. M.D.D.M., Dr. N. Muthuveeran. M.D.D.M., Dr. R. Manivannan. M.D.D.M., Dr. P.K. Murugan, M.D.D.M., Dr. R. Kishore. M.D.D.M., and Dr. C. Justin. M.D.D.M., for their valuable suggestions and support.

I owe my sincere thanks to all those patients and the technical staffs who participated in the study for it is their cooperation which made this study possible.

CONTENTS

S.No.	Title	Page No.
1	INTRODUCTION	1
2	REVIEW OF LITERATURE	3
3	AIM OF THE STUDY	24
4	MATERIALS AND METHODS	25
5	OBSERVATIONS	28
6	DISCUSSION	38
7	RESULTS	49
8	CONCLUSION	51
9	REFERENCES	
10	PROFORMA	
11	MASTER CHART	

INTRODUCTION

Seizures have been recognized since antiquity. One of the earliest descriptions of a secondarily generalized tonic-clonic seizure was recorded over 3000 years ago in Mesopotamia, which was attributed to the god of the moon. Epileptic seizures were described in ancient cultures including those of China, Egypt, and India.

The word seizure is derived from Latin word "sacire", meaning, "to take possession of" indicating that the person having a seizure is possessed or atleast out of control. The clinical symptoms in seizures could be motor, sensory, autonomic, or psychic events although in practice, when a patient presents to a health care system with a seizure it is usually a convulsive (motor) seizure, either generalized or focal.

Cumulative observations of many clinical investigators, along with adjunctive neurophysiological, imaging and genetic tools created a well-accepted diversity in the etiologies of seizures in various age groups. Interestingly, the highest incidence of seizures occur in early childhood and late adulthood. In older adults and elderly the frequent causes are cerebrovascular disease, brain tumors, alcohol withdrawal, metabolic disorders , degenerative diseases and idiopathic.

With aging population growing in our nation, thanks to improved medical infrastructure, a growing number of elderly people will be seeking attention to our health care system. This study is to analyze the seizures occurring in age group more than 50 years.

REVIEW OF LITERATURE

Historical aspect

Basic concepts surrounding seizures and epilepsy are found in ancient Indian medicine which dates back to Vedic period of 4500-1500BC. In the Ayurvedic literature of Charaka Samhita , seizures were identified and epilepsy was described as "*apasmara*" which means "*loss of consciousness*".

The foundation of our modern understanding of the derangement of function seen in seizures and epilepsy was laid in the 19th Century with the work of Hughlings Jackson. Working in Germany during the 1920s, Hans Berger, a psychiatrist, developed the human electroencephalograph. Another recent stimulus towards the understanding and treatment of seizures in the last few decades has been the development in neuroimaging. Such technology has revealed many of the more subtle brain lesions responsible for seizures.

Background:

Many diseases can cause paroxysmal clinical events. The correct diagnosis of the paroxysmal event is necessary to provide correct treatment. If the event is an

epileptic seizure, the seizure type and associated clinical, electroencephalographic (EEG), and neuroimaging findings assist in determining the risk of seizure recurrence and the possible need to begin anticonvulsant therapy

Definitions of Terms

[as per the [International League Against Epilepsy \(ILAE\) Guidelines](#) (Jallon, 1993; Roger, 1989).]

A **nonepileptic event** is a clinical event presumed to be unrelated to abnormal and excessive neuronal discharge. An example of a nonepileptic event is syncope.

An **epileptic seizure** is a clinical event presumed to result from an abnormal and excessive neuronal discharge. The clinical symptoms are paroxysmal and may include impaired consciousness and motor, sensory, autonomic, or psychic events perceived by the subject or an observer.

A **provoked seizure** is an acute symptomatic seizure that occurs following a recent acute disorder such as a metabolic insult, toxic insult, CNS infection, stroke, brain trauma, cerebral hemorrhage, medication toxicity, alcohol withdrawal, or drug withdrawal.

An **unprovoked seizure** is a cryptogenic or a remote symptomatic seizure.

Epilepsy occurs when 2 or more epileptic seizures occur unprovoked by any immediately identifiable cause. The seizures must occur more than 24 hours apart. In epidemiologic studies, an episode of status epilepticus is considered a single seizure. Febrile seizures and neonatal seizures are excluded from this category.

Frequency:

A first seizure is a sudden frightening event for the individual, onlookers and family members. Available data. on an individual's lifetime risk of developing one episode of non-febrile seizures is atleast 4% (THE LANCET. vol 352 . sep 26,1998) A first seizure caused by an acute disturbance in brain function (acute symptomatic or provoked) is unlikely to recur (3 to 10 %). If a seizure is unprovoked, however, meta-analysis suggests that 30 to 50 % will recur; and after a second unprovoked seizure, 70 to 80 % recur, justifying the diagnosis of epilepsy. (BMJ vol 332 11 feb 2006)

Race:

Racial differences have not been studied.

Sex:

Most authors

report a small-to-moderate increased preponderance in men in their studies of first seizures in adults (van Donselaar, 1992; Musicco, 1997; Hopkins 1988; King 1998).

Age

In practice it is useful to consider the etiologies of seizures based on age of the patient, as age is one of the most important factors determining both the incidence and likely causes of seizures.

Causes for seizures in Neonates:

- Perinatal Hypoxia and Ischemia.
- Intracranial Hemorrhage and trauma.
- Acute CNS Infection.
- Metabolic disturbances.
- Drug Withdrawal.
- Genetic Disorders.

Causes for seizures in Infants and Children:

- Febrile seizures.
- Genetic Disorders
- Developmental Disorders.
- Trauma.
- Idiopathic.

Causes for seizures in Adolescents:

- Trauma.
- Genetic Disorders.
- Infection.
- Tumor.
- Illicit drugs.
- Idiopathic

Causes for seizures in young adults:

- Trauma.
- Alcohol Withdrawal.
- Illicit drug use.
- Tumor.
- Idiopathic.

Causes for seizures in older adults:

- Cerebrovascular disease

- Brain tumor
- Alcohol withdrawal
- Metabolic disorders
- Degenerative diseases
- Idiopathic

CLASSIFICATION OF SEIZURES

Seizures are divided into two broad categories--generalized and partial. Generalized seizures arise from both sides of the brain simultaneously. Partial (ie, focal) seizures occur within one or more restricted regions of the brain and are a secondary effect of a localized physiologic or structural abnormality of the brain (eg, tumor, dysplasia, stroke, trauma).

International League Against Epilepsy Revised Classification of Epileptic Seizures

1. Partial (focal, local) seizures:
 - A. Simple - motor, somatosensory, autonomic, psychic
 - B. Complex
 - A. Impaired consciousness at outset
 - B. Simple partial followed by impaired consciousness
 - C. Partial seizures generalized tonic-clonic (GTCS)

A. Simple to GTCS

B. Complex to GTCS

2. Generalized seizures (convulsive or non-convulsive)

A. a. Absence seizures

b. Atypical absences

B. Myoclonic

C. Clonic

D. Tonic

E. Tonic-clonic

F. Atonic

G. Combinations

3. Special epileptic seizures

Myoclonic seizures

Reflex Epilepsy

Acquired aphasia with convulsive disorder

Febrile seizures

Hysterical seizure

Evaluation of a first seizure

The first step in evaluating a suspected seizure is to determine whether the event was, in fact, a seizure

Syncope is often mistaken for an epileptic seizure . During many syncopal episodes, clonic or myoclonic jerks occur in the distal portions of the extremities. Convulsive syncope occurs when there is severe or prolonged reduction of blood flow to the brain, resulting in an event resembling tonic-clonic seizure. Diagnosis is based on possible provocative factors in the medical history (eg, pain, dehydration), physical examination (eg, orthostatic blood pressure check), and studies such as electrocardiography and tilt table testing .

Another common imitator of epileptic seizure is the nonepileptic psychogenic seizure. No single feature reliably differentiates the two disorders. However, many ictal features of nonepileptic psychogenic seizure are uncommon in epileptic seizures. For example, such features as gradual onset, stopping and restarting of motor activity, out-of-phase clonic movements of the extremities, vocalization in the middle of the seizure rather than at the start, pelvic thrusting, and lack of body rigidity are more common in psychogenic seizures than in tonic-clonic seizures In addition, the typical duration of a tonic-clonic seizure is 50 to 92 seconds, whereas the range for psychogenic seizures is 20 to 805 seconds.

Furthermore, some epileptic seizures have symptoms that are frequently misdiagnosed as psychogenic. Frontal lobe complex partial seizures often last less than 1 minute and sometimes include rocking, kicking, "bicycling," pelvic thrusting, genital manipulation, and cursing. Their lack of postictal symptoms also makes frontal lobe seizures difficult to differentiate from psychogenic seizures.

Other NonEpileptic Paroxysmal Events:

Migraine (classic [with auras], basilar, confusional)

Cerebrovascular event (transient ischemic attack)

Periodic paralysis

Sleep disorders (parasomnias, daytime amnestic episodes)

Gastrointestinal disorders (reflux, motility disorders)

Movement disorders (tics, Tourette's syndrome, nonepileptic myoclonus, paroxysmal choreoathetosis, shuddering attacks)

Psychiatric disorders (panic, somatization, dissociation, conversion [nonepileptic psychogenic seizures])

Drug toxicity and substance abuse

Breath-holding spells

History of the event

A description of the circumstances surrounding a paroxysmal event can provide important diagnostic clues. A witnessed, 90-second episode that involved loss of consciousness, stiffening, and jerking of the extremities followed by muscle soreness, headache, and the need to sleep for several hours afterwards strongly suggests a tonic-clonic seizure

KEY ELEMENTS IN HISTORY

Before the event

Unusual stress (eg, severe emotional trauma)

Sleep deprivation

Recent illness

Unusual stimuli (eg, flickering lights)

Use of medications and drugs

Activity immediately before event (eg, change in posture, exercise)

During the event

Symptoms at onset (eg, aura)

Temporal mode of onset: gradual versus sudden

Duration: brief (ictal phase <5 min) versus prolonged

Stereotypy: duration and features of episodes nearly identical versus frequently changing

Time of day: related to sleep or occurring on awakening

Ability to talk and respond appropriately

Ability to comprehend

Ability to recall events during the seizure

Abnormal movements of the eyes, mouth, face, head, arms, and legs

Bowel or bladder incontinence

Bodily injury

After the event

Confusion

Lethargy

Abnormal speech

Focal weakness or sensory loss (ie, Todd's paralysis)

Headache, muscle soreness, or physical injury

Past medical history

A review of the events leading up to the seizure may reveal factors that suggest it was provoked. Causes of provoked seizures include alcohol withdrawal, substance abuse, hypoxia, fever, electrolyte imbalance, hypoglycemia, and sleep deprivation.

Drug history

Theophylline, meperidine hydrochloride, isoniazid, antipsychotic drugs (especially clozapine and phenothiazines), radiocontrast dyes, alkylating agents, and β -lactam antibiotics, quinolones are among the most commonly implicated medications in seizure.

However, many other drugs can cause seizure, including lidocaine hydrochloride, general anesthetics, tricyclic antidepressants, selective serotonin reuptake inhibitors, bupropion hydrochloride, acyclovir, β -blockers, and decongestants (eg, phenylpropanolamine hydrochloride). Also, seizures can be provoked by alcohol withdrawal as well as use of cocaine.

Physical examination

A thorough physical examination can help uncover possible causes of a seizure. Findings may include evidence of trauma, infection, malignancy, congenital anomalies,

and prior neurologic events (eg, focal weakness, spasticity suggesting previous stroke).

During an emergency department evaluation of a patient immediately after a seizure, vital signs should be measured and a general medical examination performed. Guidelines for physical examination are as follows:

- Examine the patient for injuries from the seizure or fall.
- Check oxygen saturation and auscultate the chest for possible aspiration.
- Measure heart rhythm and rate, blood pressure, and orthostatic changes for assessment of syncope.
- Auscultate for carotid murmurs or carotid bruits and sources of embolic stroke.
- Check for rapid pulses, which are often present after seizure and may help in evaluation of psychogenic seizures.

An electrocardiogram should be obtained to identify cardiac rhythm, detect possible ischemia, and measure the QT interval. Prolonged QT syndrome often presents with simple or convulsive syncope. Electrocardiography and 24-hour ambulatory continuous electrocardiographic (Holter) monitoring can help identify cardiac arrhythmias. The possibility of a recent myocardial infarction should be considered, particularly in elderly patients, in whom myocardial infarction may occur from the stress of a seizure.

Neurologic examination

The purpose of the neurologic examination is to identify focal or diffuse cerebral dysfunction. This information is particularly helpful in localization-related epilepsy. The presence of various features offers clues to the focus of a seizure. For example, aphasia suggests a left frontal, temporal, or parietal onset. Right or left hemiparesis suggests foci from the contralateral motor cortex.

In initial evaluation of a seizure, patients should be observed for fluency of language, facial asymmetry, gaze preferences, and pupillary asymmetry. The last presents in patients who have herniation from brain swelling caused by parenchymal or epidural bleeding and in those who have a rapidly growing brain tumor. The presence of pronator drift may indicate subtle weakness not detected by strength testing. Sensory deficits suggest parietal lobe dysfunction. An extensor plantar response may be noted for some time after a seizure and is not necessarily a pathologic finding.

Diagnostic testing

Laboratory workup is an essential part of evaluation of seizure. Measurement of glucose, calcium, magnesium, thyroid hormone, and liver enzyme levels, as well as toxicology screening (including blood alcohol levels), may reveal common medical causes of seizures. A complete blood cell count may suggest infection, anemia, or sickle cell disease.

In patients suspected to have had an infection or a fever or to have exhibited abnormal behavior just before the event, lumbar puncture should be performed after assessment of the possible risks of the procedure (eg, coagulopathy, mass lesion). Patients who are immunocompromised because of corticosteroid use, recent transplantation, or HIV infection should undergo cerebrospinal fluid evaluation to detect possible fungal, bacterial, or viral infection. In patients with a systemic malignant condition, cytologic evaluation of cerebrospinal fluid can identify meningeal carcinoma.

Electroencephalogram:

- EEG should be performed within 24 hours of the seizure because it is significantly more sensitive when obtained during that period (King, 1998). If the routine EEG findings are normal, a sleep-deprived EEG should be performed.
- Standard EEG detects epileptiform discharges in 29% of patients. Standard EEG combined with sleep-deprived EEG shows epileptiform discharges in 48% of patients (van Donselaar, 1992).
- Schreiner and Pohlman-Eden studied the value of an EEG taken within 48 hours of the first seizure in an adult. They found that 38.0% of patients without seizure recurrence had normal EEGs, while only 10.2% of patients with seizure recurrence had normal EEGs. Focal epileptiform activities were found

significantly more frequently (26.5% vs. 13.0%) in patients with seizure recurrence than in patients without seizure recurrence.

Limitation of EEG:

An

estimated 0.4% of adults and 2.8% of children who have never had a seizure may have interictal epileptiform discharges. Furthermore, a normal EEG does not refute the diagnosis of epilepsy. The initial EEG reveals epileptiform activity in only 40% of the patients with probable epilepsy.

Imaging studies

The role of imaging studies depends on the stage of evaluation. Immediately after a seizure, computed tomography can detect the presence of bleeding or gross structural lesions. However, magnetic resonance imaging is the study of choice because it is more sensitive and specific for evaluating structural lesions and brain parenchyma. Particular attention should be directed to the hippocampus for evaluation of lesions (eg, mesial temporal sclerosis) and to the cortical architecture for detection of abnormalities (eg, dysplasia).

Are Antiepileptic drugs needed after a first seizure?

Drug treatment after first seizure is controversial. Too large recent randomized

studies of children and adults compared antiepileptic drugs with, no treatment after a first seizure and came to an identical conclusion. Any decision to start treatment must weigh the risk of another seizure against the risks of side effects from chronic drug treatment.

Risk factors for recurrent seizures include the following

- Age younger than 16 years: Musicco et al found that children younger than 16 years had almost double the risk of recurrent seizures as adolescents and adults aged 16-60 years (Musicco, 1993).
- Remote symptomatic seizure (Annegers, 1986; Hauser, 1990; Berg, 1991): In the case of seizures after a first stroke, Labovitz et al found that lesion location and stroke subtype are strong predictors of early seizure risk, and early seizures are a predictor of recurrent seizures (Labovitz, 2001).
- Seizures occurring between midnight and 8:59 am (Hopkins, 1988; Martinovic, 1997; Bora, 1995)
- Prior provoked seizures (Hauser, 1990)
- Remote symptomatic seizure in a patient whose sibling is affected with epilepsy (Hauser, 1990)
- Status epilepticus or multiple seizures within 24 hours as the initial remote symptomatic seizure (Hauser, 1990)

- Partial seizures (Annegers, 1986; Berg, 1991)
- Todd paralysis in patients with a remote symptomatic seizure (Hauser, 1990)
- History of neurological deficit from birth such as cerebral palsy or mental retardation (Annegers, 1986)
- Abnormal examination findings in patients without a remote symptomatic seizure (Annegers, 1986; Camfield, 1985)
- CT scan that shows a brain tumor (Hopkins, 1988)
- EEG that shows epileptiform discharges
 - In patients with a first seizure and no known etiology, van Donselaar obtained a routine EEG in all cases and a second sleep-deprived EEG if the first EEG did not show epileptiform discharges. His pooled results showed the following 2-year cumulative risks of seizure recurrence: in patients with epileptiform discharges, 83%; in patients with nonepileptiform abnormalities, 41%; and in patients with normal EEGs, 12% (van Donselaar, 1992).
 - In 1997, Beghi et al found that epileptiform discharges were associated with a 1.5- to 3-fold increase in the risk of seizure recurrence.
 - In 1993, Musicco et al found that epileptiform discharges were associated with a 1.7-fold increased seizure recurrence risk.
 - Berg and Shinnar found that epileptiform discharges were associated with a 2-fold increased seizure recurrence risk (Berg, 1991).
 - In 1990, Hauser et al found that generalized spike and wave increased the risk of

recurrent seizure in patients with no known etiology.

- In 1997, Beghi et al found that an abnormal EEG finding and the presence of an underlying etiology (remote symptomatic) are the most consistent predictors of recurrence.

If drug treatment is considered , which drug is preferred ?

If drug treatment is considered after first seizure, the chosen antiepileptic drug should have high efficacy, good tolerability and low interaction potential and allow a good quality of life, especially since half of all patients would never have another seizure without treatment. The starting dose should be in the lower range

If an underlying epilepsy syndrome has been established, the following antiepileptic drugs are available³.

	GTCS	Partial	Absence	Myoclonic,
First	Valproic Acid	Carbamazepine	Valproic Acid	Valproic Acid
Line	Phenytoin	Phenytoin	Ethosuximide	
	Carbamazepine			
Alternatives	Lamotrigine	Oxcarbazin	Lamotrigine	Lamotigine
	Oxcarbazin	Lamotrigine	Clonazepam	Topiramate

		Levetiracetam		
--	--	---------------	--	--

Further Inpatient Care:

- Many patients who have a seizure recover spontaneously and fully with normal consciousness after a short time interval. Patients with incomplete recovery or a prolonged postictal state may require inpatient hospitalization (Moore-Sledge, 1997).
- Inpatient management may be necessary if the clinical course is complicated by other medical problems requiring inpatient management.

A short hospitalization may be necessary for patients who are at risk of recurrent seizures and have no adequate supervision at home. Patients admitted from an emergency department had a 16.8% risk of an early recurrent seizure during their brief hospitalization (Tardy, 1995). This risk of early recurrent seizures was higher than reported in other studies (Hauser, 1990; Musicco, 1993; Annegers, 1986).

Summary

The first step in evaluation of a presumed seizure is to determine whether the event was indeed a seizure and which diagnostic studies are needed. The second step is to correctly diagnose the seizure on the basis of the medical history and findings from

the physical, neurologic, and laboratory evaluation. The third step is to decide whether drug treatment is necessary. Every paroxysmal event is unique, and not every seizure needs to be treated. When treatment is deemed appropriate, an antiepileptic drug should be chosen after consideration of the risk-benefit profile of the available agents.

AIM OF THE STUDY

- To study the etiologic profiles of first episode seizures in patients aged more than 50 years of age.
- To analyze the age / sex distribution, presenting history, clinical findings and investigations at admission in the study group.

MATERIALS AND METHODS

The study was done in the setting of the Department of Neurology and Neuro Surgery, Govt. Rajaji Hospital, Madurai. The study had collaborations with the Department of Medicine, Biochemistry, Pathology, Radiology and Microbiology.

The study was observational in nature designed to analyze patients in age group more than 50 years of age and who presented with first onset seizures. The sample size was 82 and the study period was from January 2007 to December 2007.

INCLUSION CRITERIA

- Patients who presented with first onset seizures over the age of 50 years admitted in our unit.
- Inpatients who developed first seizure in this hospital.
- Time between seizure and presentation to us within 15 days.

EXCLUSION CRITERIA

- History of trauma
- History of ingestion of toxins.

Clinical data was collected from patients and witnesses in a systematic manner

and added to a database, which included a checklist of seizure antecedents and the symptoms associated with seizure.

The first task was to ascertain if at all, the presenting complaint is a seizure. In a few instances, even when the presenting history was ambiguous seizure recurrences were witnessed for confirmation. The clinical diagnosis on the seizure type, whether partial or generalized was made.

In-depth probes in the history for provocation factors and features suggesting organicity were attempted. Significant past medical history if any were noted. A thorough clinical examination was performed at the time of admission and relevant findings recorded. A routine metabolic screening, which included blood sugar, urea, serum creatinine, electrolytes and liver function tests (if indicated), were done at the time of admission.

Lumbar puncture and CSF analysis was done if infective etiologies were suspected.

Earliest possible EEG was attempted and was performed using 32 channel digital EEG recorder.

CT brain plain study in all patients and contrast studies when necessary were done in all patients in the study group. MRI brain was done when indicated.

Limitations were encountered in affordability of patients for MRI scanning. Early EEG (within 24hours of onset of seizures) could not be performed due to delay in referral of the patients to this institution and because of the time taken for stabilizing patients. EEG could not be done in a some cases owing to emergency surgical interventions.

OBSERVATION

Seizures in 82 patients in the age group of 50 to 82 are studied; of which 50 are males and 32 are females.

Table 1 sex distribution of patients

Sex	number	percentage
Male	50	61%
female	32	39%

Table 2 shows the distribution of various age group in the study.

Age group	percentage
50 – 60	46%
60 – 70	44%
70 – 80	08%
> 80	02%

In the study group, 82% of patients had prior consultations/ treatment elsewhere before reaching this hospital.

Table 3 The nature of health care system patients first seeked after the first episode of seizure.

Private practitioners	37%
Private hospitals	26%
Government institutions	13%
Directly reached	12%

The seizures are grouped as per international league against epilepsy-revised classification of epileptic seizures as partial seizures and generalized seizures. (A partial seizure with secondary generalization was found in 10% of the study group).

Table 4 seizure types found in the study

Seizure type	Number of patients (%)
Generalized	58 (71%)
partial	24 (29%)

Table 5 coexistent non convulsive symptoms at admission

Symptoms	Frequency
Limb weakness	68%
Headache	43%
Fever	25%
Vomiting	21%
Inability to talk	21%
Visual disturbances	15%

Table 6 observation in history for organicity of seizures

History suggestive of organicity	Frequency
Post ictal confusion	63%
Frothing of saliva	38%
Seizures during sleep	21%
Bladder / bowel incontinence	19%
Tongue biting	04%
Bodily injuries	04%

Table 7 - Profile of significant past medical history

Past medical history	No of patients
Diabetes	08
Hypertension	11
Both diabetes and hypertension	03
Renal failure	02
Liver disease	01
Pulmonary tuberculosis	03
Heart disease	06

During an emergency evaluation of patients at admission vital signs were monitored which revealed 16% of patients were hemodynamically unstable.

Neurological examination revealed abnormality in 68% of patients.

Table 8 Spectrum of neurological signs at admission.

Motor system abnormalities	48%
Altered sensorium	39%
Signs of meningeal irritation	19%
Cranial nerve abnormalities	13%
Status epilepticus	03%
Cerebellar signs	02%

Metabolic abnormalities at the time of admission were investigated, as they are among the most readily treatable causes of seizures. The abnormalities in metabolic parameters were noted in 38% of patients in this study.

Table 9 Metabolic abnormalities in patients at admission

Metabolic abnormality	Number of patients
Hyponatremia	12
Hyperglycemia	11
Metabolic Acidosis	06
Hypoglycemia	04
Renal failure	03

Total patients	Number of patients in whom EEG was done (%)	Number of Abnormal records In whom EEG was done (%)
82	68 (83%)	56 (68%)

Lumbar puncture and CSF analysis done in 14 patients suspected of meningitis or encephalitis revealed abnormality in five cases.

EEG was taken after stabilizing the patient and all were taken in the inter ictal period. EEG was done in 68 of the 82 patients (83%) in this study. Abnormalities were found in 56 of the 68 patients subjected to EEG.

The most common observed pattern in EEG was Sharp & Spike waves, Poly spike pattern during the inter ictal period.

Table 10 : EEG in this study

Table 11

	Generalised	Focal
Sharp & Spike waves	28	8
Poly spikes		
Diffuse slowing	8	12

EEG patterns observed were

Normal, Diffuse slowing, Sharp discharges, Focal spike, Asymmetrical narrowing, Polyspikes and others.

When the other investigations were inconclusive, ‘focal findings in the EEG originating from the temporal lobes’ were recorded in two patients, which helped in the diagnosis of encephalitis.

Table 12 shows CT abnormalities in the study group

Cortical atrophy was found in combinations with various other findings. Radiologist opinion was	CT findings	Number(%)
	Cortical atrophy	22 (27%)
	Infarct	14 (16%)
	Parenchymal hemorrhage	04 (6%)
	Sub dural hemorrhage	02(03%)
	Tumors	11 (13%)
	Ring enhancing lesions	03 (04%)

obtained on all CT scans. CT scan brain was done in all the patients in the study group.

MRI scanning of brain could be done only in 15 patients of the study group. Besides improvement in details of CT findings MRI was helpful in uncovering lesions missed in CT.

Table 13 New lesions uncovered in MRI

New lesion uncovered in MRI	Number
Tumors	02
Arterial Infarcts	02
Encephalitis	02
Cerebral venous thrombosis	02
Granuloma	01

Table 14 Etiology profiles in the study group

with mean age of distribution

ETIOLOGY	TOTAL		
	PATIENTS	%	MEAN AGE
CVA	22	26.82	60
TUMORS	13	15.85	63
METABOLIC	11	12.19	61
ALCOHOL WITHDRAWAL	08	9.76	58
GRANULOMA	04	04.88	51
ENCEPHALITIS	05	06.08	59
CVT	02	02.43	63
MENINGITIS	03	03.66	52
SUBDURAL HEMORRAGE	02	02.43	67
UNIDENTIFIABLE	12	13.41	61

Four of the 82 patients presented with status epilepticus. At least one recurrence in the month following first seizure was noted in 31 (46%) patients. In hospital death occurred in two patients who were admitted for first seizures.

DISCUSSION

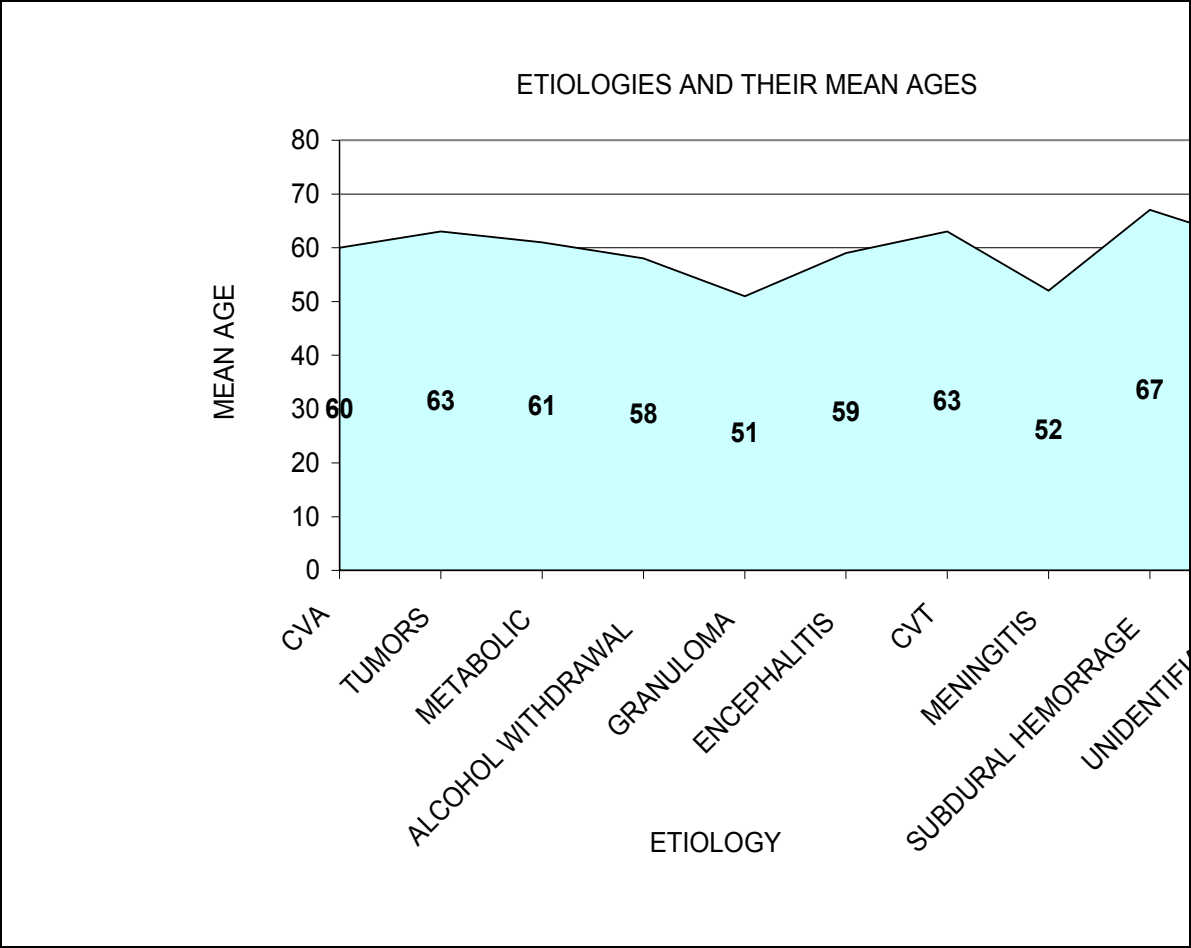
The study group comprised of 62% males and 38% females. Most authors report a small-to-moderate preponderance of men in their studies of first seizures in adults (van Donselaar, 1992; Musicco, 1997; Hopkins, 1988; King, 1998). A male to female ratio of **1.6: 1** is observed in this study, a trend noted in other studies.

Analyzing the age groups in this study the maximum incidence of first onset of seizures is found in the age interval of 60 to 70 years. Studies have shown that incidence of new onset seizures above age 65 is even higher than first year of life – 135 per 100000 vs. 79 per 100000 .

The mean age for most of the common etiologies in this study was around 61 and hence, the age of a given patient with new onset seizure does not favor any particular etiology. The less common etiologies in this study, granulomas and meningitis however occurred around 51 years of age. The highest mean age encountered in this study was for sub dural hemorrhage.

After the episode of the first seizure, the nature of health care system first sought by the patient when analyzed showed that 63% sought to hospitals, as against 37% who sought to private practitioners.

ETIOLOGIES AND THEIR MEAN AGES



This is a trend towards more patients seeking institutionalized care after a first onset seizure.

The seizure type classified in this study as per International League Against Epilepsy-revised classification of epileptic seizures revealed generalized seizure in 71% and partial seizure in 29%.

Zhu PG studied new onset seizures in the ages between 20 and 80 revealed generalized seizures in 64% and partial in 30%.

Retrospective study of Perez et al in 250 patients with late onset seizures revealed 59% generalized and 41% partial in nature.

The observation of seizure types in this study is almost similar to the above-mentioned studies.

**Table 15 Comparison of seizure type encountered in this study
with various studies.**

Seizure type	This study	Zhu PG	Perez et al
Generalized	71%	64%	59%
partial	29%	30%	41%

The seizure typing in this study was entirely made with history. The grey area in relying on history in classification of seizures is in the fact that the focal onset of a seizure is often missed and witnesses attention is often drawn to the person only after an event becomes generalized.

Limb weakness and headache were the most common non-convulsive symptom, which the patients / attenders complained at admission. Fever presented at admission in 25% of patients in this study. It is important here to reemphasize that fever is one of the provoking factors for seizures. Vomiting presented in 90% at the time of seizures in patients with tumors. Papiloedema was present in 20% of patients with tumors.

Post ictal confusion was the most frequent factor present in the history to suggest organicity.

A history of alcohol intake, in most of the days of a week for more than ten years was present in 20% of patients.

Previous history of diabetes was present in 11 patients, two of whom presented with hypoglycemic seizures. Both of them were on glibenclamide and one of them was found to have renal failure. One patient presented with seizures associated with a nonketotic hyper osmolar state.

Motor system abnormalities and altered sensorium were the most common neurological signs present at the time of admission. It was the motor system abnormality in clinical examination, which most frequently predicted an abnormality in the CT scan.

Of the three patients admitted with status epilepticus two were found to have tumors and one had alcohol withdrawal seizures. Signs of meningeal irritation were present in four patients with tumor etiologies and three patients with CVA besides presenting in all patients with meningitis. Sixth and seventh were cranial nerves comprised most of the cranial nerve abnormalities. The patient who presented with cerebellar signs was later found to have posterior circulation stroke

Metabolic abnormalities contributed to etiology in 13% of patients and most of them were readily treatable, hence a thorough search for these factors should be the early priorities. The most common metabolic abnormality was hyponatremia which was often encountered as an associated finding with other etiologies.

The study newly detected eight patients to have Type 2 diabetes, 11 to have hypertension and four to have both. Renal failure was detected in eight of the patients in the group.

EEG was done in 39(57%) of the 68 patients in the study. Abnormalities were found in 18(46%) of the EEG's done. The average period from the onset of seizure to the record of EEG was six days, owing to the late referral of patients to this institution and to the time taken to stabilize the patient before shifting to EEG room. The yield of

abnormalities in the EEG in this study could have been better if it were done more early or special methods such as continuous EEGs and sleep deprived EEGs were adopted.

The most common abnormality in EEG was Sharp & Spike waves and poly spike activity. Anti-convulsant drugs slow the normal background rhythm in EEG and almost 80% of the patients in the study group were under the anti convulsant drugs when EEG was performed, which explains the predominance of diffuse slowing pattern in the EEG.

When the other investigations were inconclusive, “focal findings in the EEG originating from the temporal lobes” were recorded in two patients, which helped in the diagnosis of encephalitis.

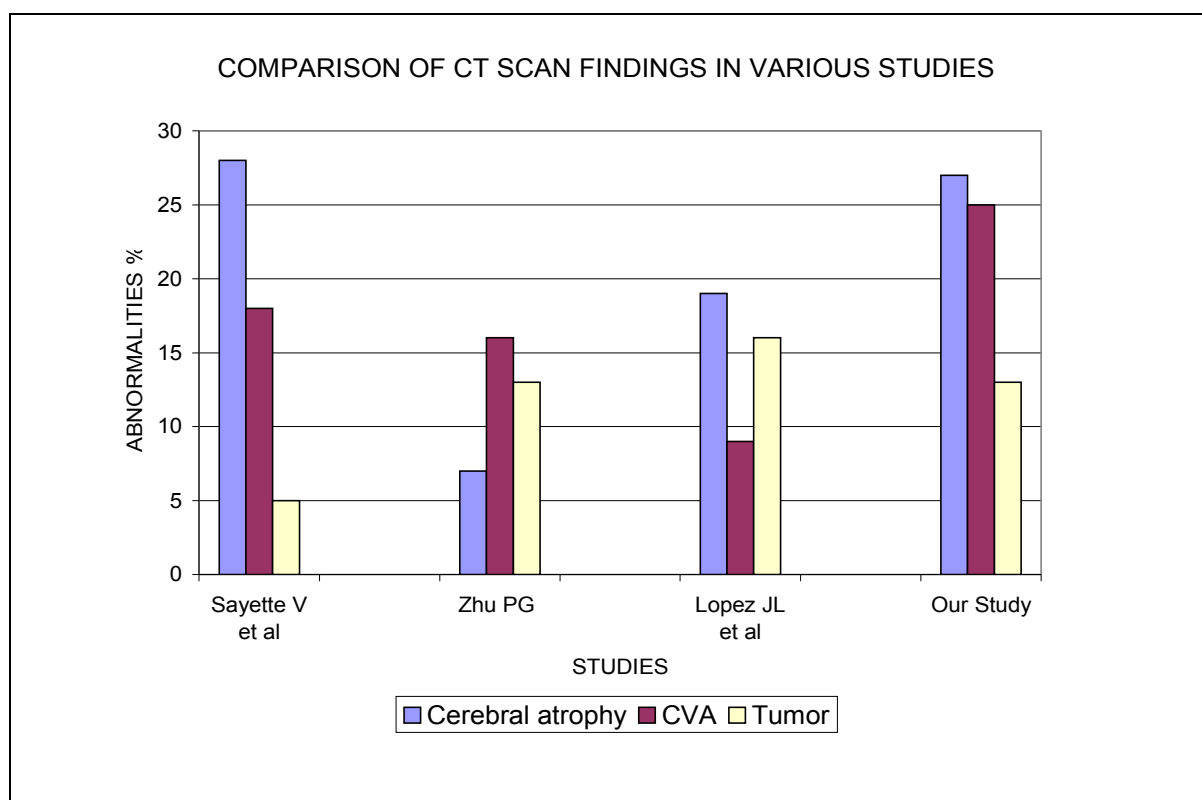
CT scan was done in all patients in the study group, in which the abnormalities contributed to the etiologies in 43% of patients. Cerebral atrophy (27%) was the most common abnormality present in the scan report but had no relevance with any etiology.

Abnormal CT findings in this study included infarct (16%), tumors (13%), parenchymal hemorrhage (06%), granulomatous lesion (04%) subdural hemorrhage(03%),

CT findings in the study of new onset seizures by Sayette V et al after the age of 50 found cerebral atrophy in 29%, CVA in 75%, tumors in 5%.The spectrum of CT findings is almost similar to our study except for the low incidence of tumors.

In the study of Zhu PG, CT scan findings were compatible with CVA in 16% , tumors in 13% , atrophy in 7% and trauma in 8%. This study was done in age group ranging from 20 to 87 years. The inclusion of lesser age groups in this study explains the lower incidence of cerebral atrophy in CT scans.

Comparison of CT Scan Findings in various Studies



Affordability was a limiting factor for MRI studies in the study group. MRI despite improving the descriptions of lesions already studied in CT scans was instrumental in uncovering new lesions in nine of the patients in the study, in whom all other investigations were otherwise normal. The new lesions uncovered were tumors,

encephalitis, infarcts and CVT in two patients each and granuloma in one patient.

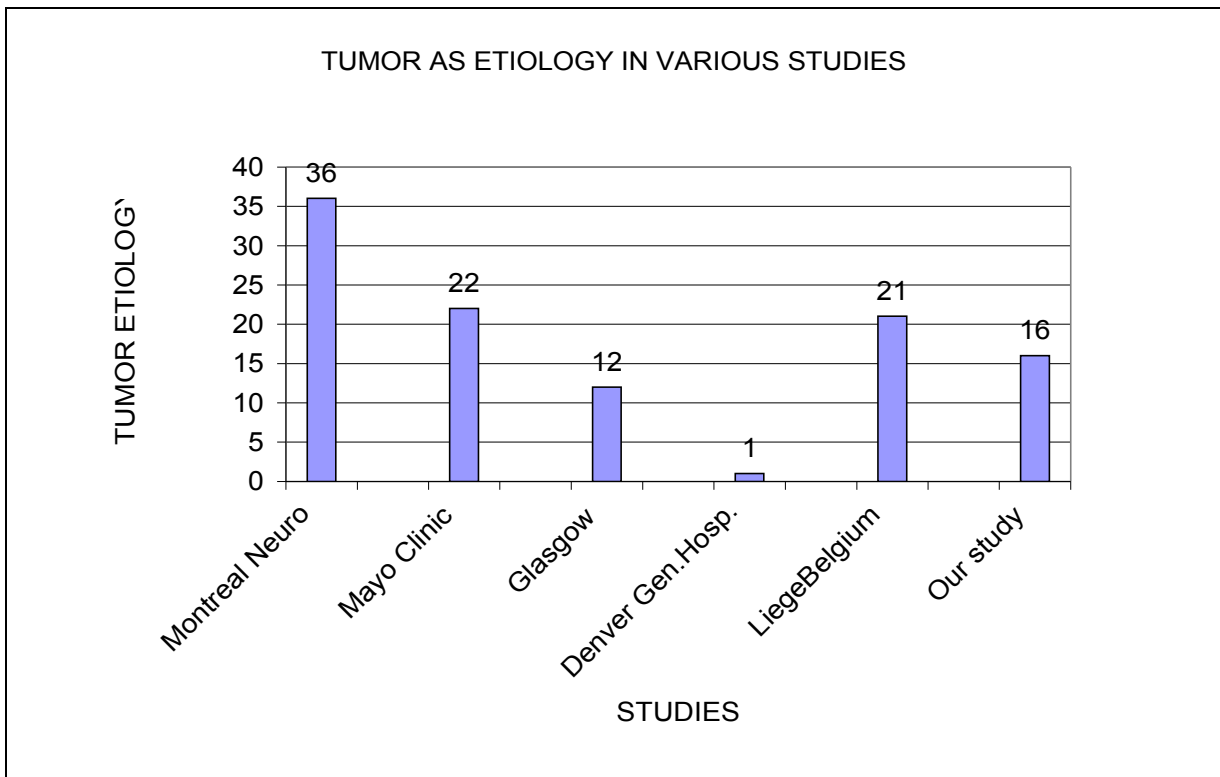
Etiologic profiles revealed CVA, tumors, metabolic causes, and alcohol withdrawal seizures contributing to 66% in this study. CVA was the single most common etiology uncovered in this study.

In the Minnesota study, the most prevalent underlying condition accounting for seizures in the elderly was stroke. Of the patients in the CVA group, 13 had infarcts and five had hemorrhages. Nine patients presented with acute CVA and seizures, whereas five had delayed post stroke seizures.

Lesser and coworkers suggested that the acute and delayed post stroke seizures have different mechanism, the former related to “transient cytotoxic metabolic alterations” and latter to structural changes, especially extravasations of blood and deposition of iron.

Tumors contributed to 16% of etiologies in this study. Of the 11 patients with tumors, eight had primary CNS tumor against three patients diagnosed to have secondaries. Tumor as etiology in various studies are as follows, Montréal neuro institute 36% (study age over 50), mayo clinic 22% (study age over 60), Liege Belgium study (age 55 to 64) 21%, Glasgow Scotland 12% (study age - elderly) and Denever general hospital 1% (study age over 69).

TUMOR AS ETIOLOGY IN VARIOUS STUDIES



Tumor as etiology in older patients lies between 1% and 36% in various studies and the result of this study (16%) lies somewhere in the midpoint of this spectrum

In 13% of patients in this study, etiology could not be determined. This significant proportion of seizures in the elderly that remain idiopathic raises the possibility that age-related changes in the brain lower seizure threshold. Animal studies are consistent with such a possibility. For example, aged rats are more susceptible to kainite-induced seizures. Also these could be the cases of late onset epilepsies.

Hyponatremia, hypoglycemia, hyperglycemia and renal failure contributed to 13% of seizures in the study. They were the most readily treatable causes, especially those patients detected to have hypoglycemic seizures. Hence a review at the metabolic parameters at admission is mandatory and when detected is most rewarding for the treating physician.

History was the tell tale evidence in patients diagnosed to have alcohol withdrawal seizures which formed 10% of etiologies.

History was the tell tale evidence in patients diagnosed to have alcohol withdrawal seizures which formed 10% of etiologies

Granulomatous etiology for first onset seizure was found in five percent of the study group. The significant point about this group was that their mean age of occurrence was the least in this study (51 years).

Cortical venous sinus thrombosis, meningitis and subdural hemorrhage in total contributed to 13% percent of the total etiology. Both the meningitis was of tuberculous etiology in this study.

Unlike seizures in the young etiologies could be established in most of the new onset seizures in age above 50 years.

Table 16 Comparison of etiological profile with young patient

ETIOLOGIES	THIS STUDY (>50 YEARS) (IN PERCENTAGE)	MINNESOTA STUDY IN YOUNG (IN PERCENTAGE)
CVA	26	1
TUMORS	16	3
IDIOPATHIC	13	83
METABOLIC	13	-
INFECTIVE	14	3
CONGENITAL	-	4

RESULTS

- The mean age of patients in the commonly encountered etiologies, in the study was around sixty years.
- The mean age of granulomatous etiology, was the least in this study.
- **Generalized seizures (71%) were the most common seizure** type encountered in the study.
- Limb weakness and headache were among the most common non-convulsive presenting symptom.
- In the clinical examination, motor system abnormality was the most consistent factor that predicted an abnormal CT scan.
- EEG, which was done in 57% of the patients in the study recorded abnormalities in 46%.
- **Cerebrovascular accidents were the most frequent etiology for the first onset seizure after fifty years of age in this study.**
- Literature reveals a great diversity in the proportions of tumors forming etiology of seizures in later ages (1% to 36%). This study established **tumors as etiology in 16% of patients.**
- CVA, tumors, metabolic causes and alcohol withdrawal formed 80% of the etiology of seizures.
- Metabolic abnormalities contributed to etiology in 13% of patients.

- CT detected abnormal lesions in 42% of cases.
- **MRI was instrumental in uncovering new lesions in nine patients.**
- In this study, causes could not be identified for 13% of the patients.

CONCLUSIONS

- **Unlike in young, most of the seizures in the age group studied, had their etiologies established. Hence, in a patient with new onset seizures more than 50 years, all efforts to identify the etiology should be made.**
- **Thorough search to rule out metabolic factors as cause of seizures should be an early priority.**
- **CT brain and MRI are indispensable in patients more than 50 years with new onset seizures.**

SEX AND AGE DISTRIBUTION OF THE PATIENTS

CHART - 1

SEX DISTRIBUTION OF PATIENTS

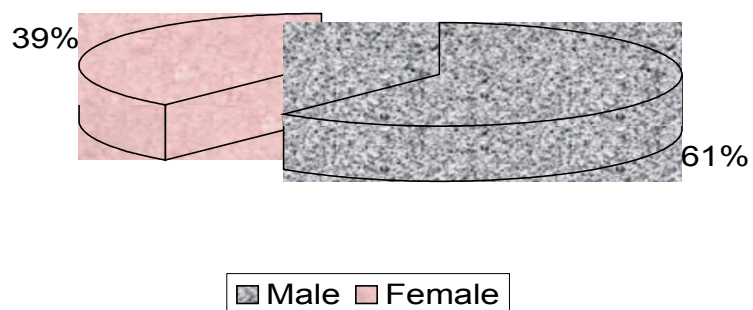
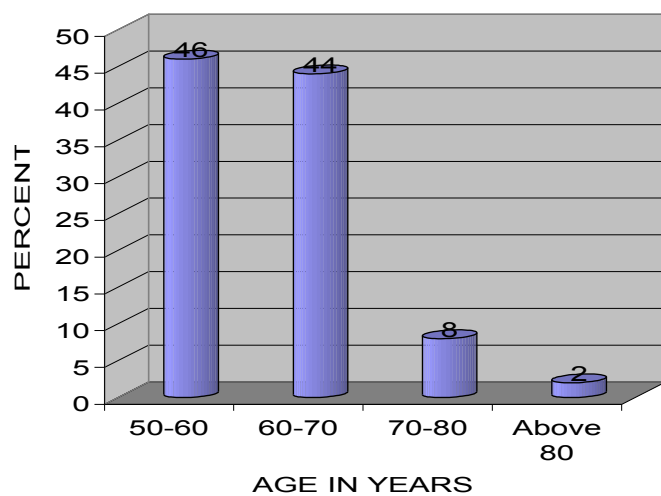


CHART-2

DISTRIBUTION OF AGE GROUP



The nature of health care system patients first seeked after the first episode of seizure

Chart 3

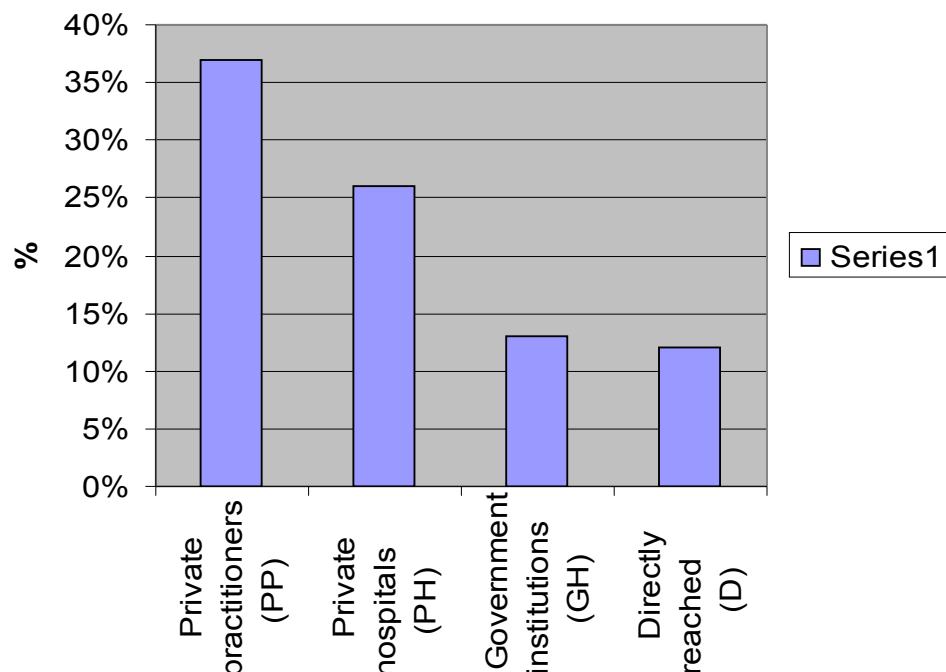


CHART -4 Seizure types found in the Study

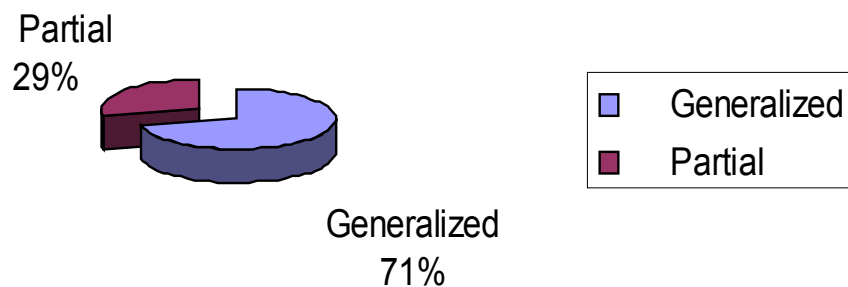


CHART -5

Coexistent non convulsive symptoms at admission

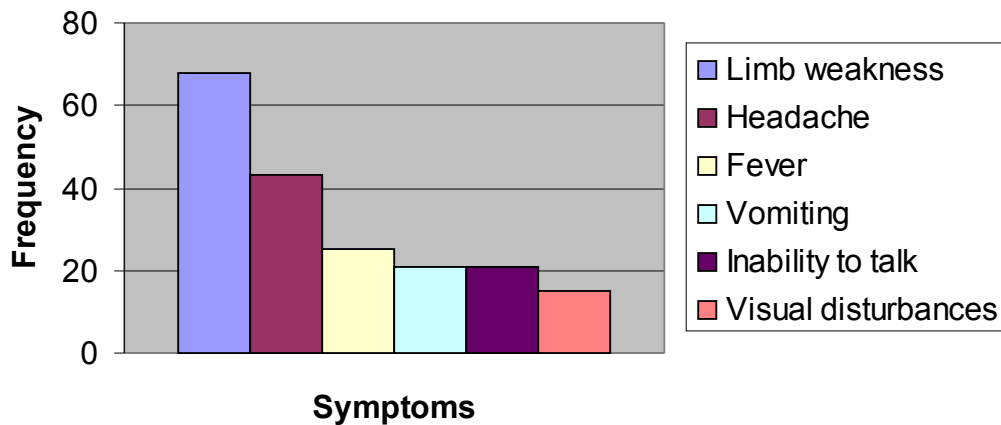
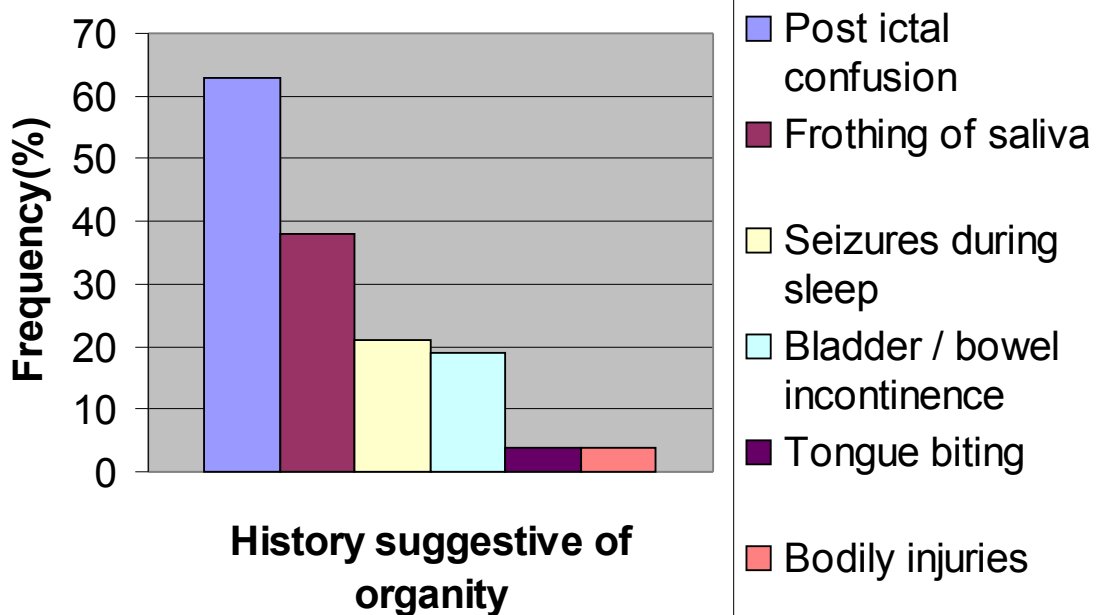


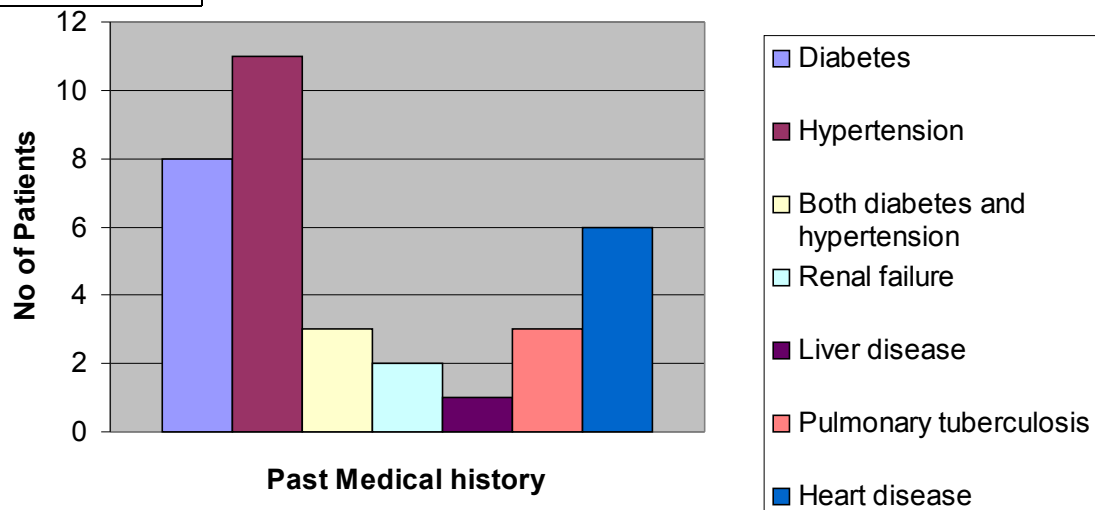
CHART -6

Observation in history for organicity of seizures



Enumerates the profiles of significant past medical history observed in this study.

Chart - 7



Spectrum of neurological signs at admission

Chart - 8

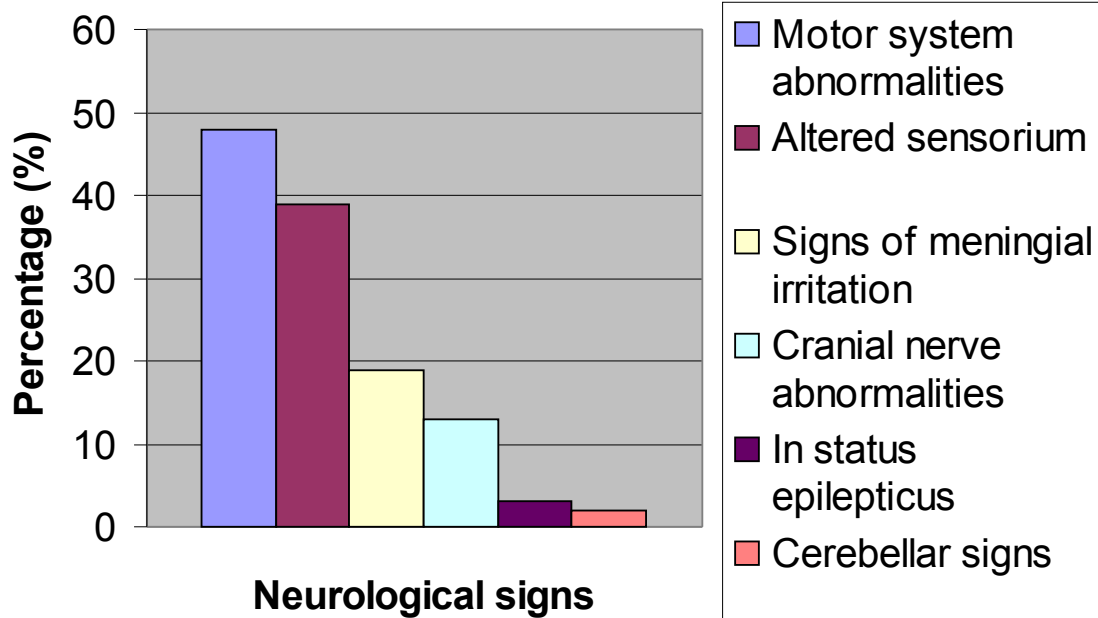
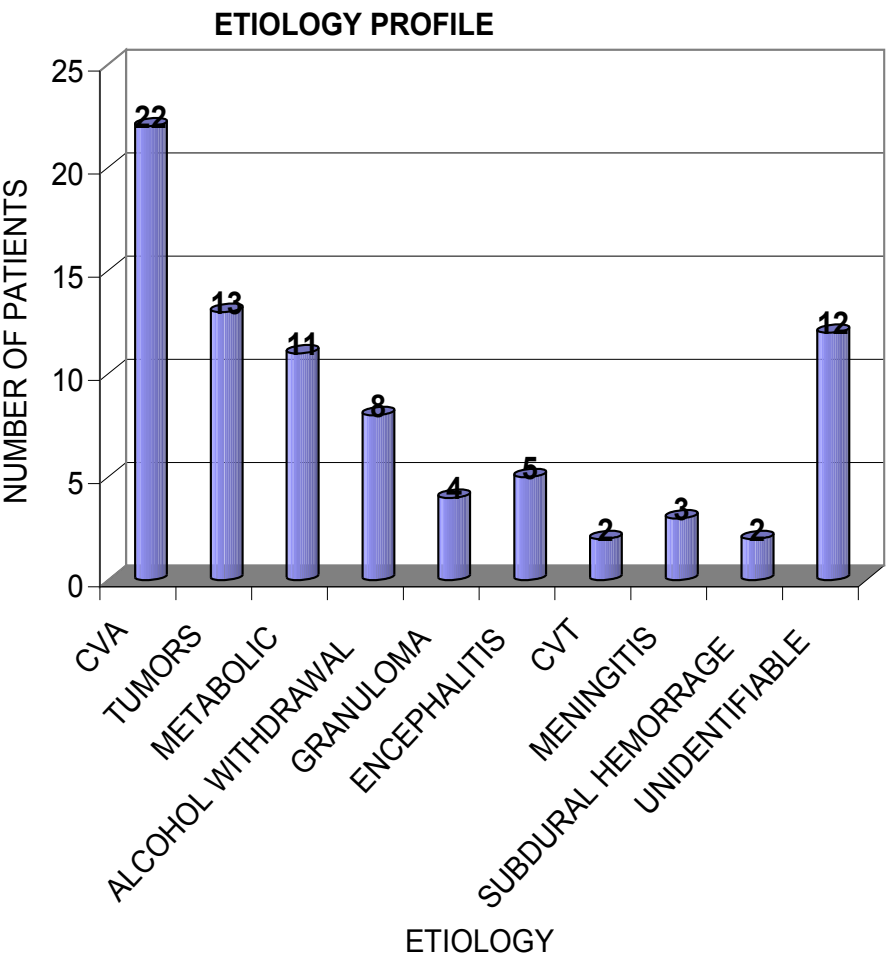
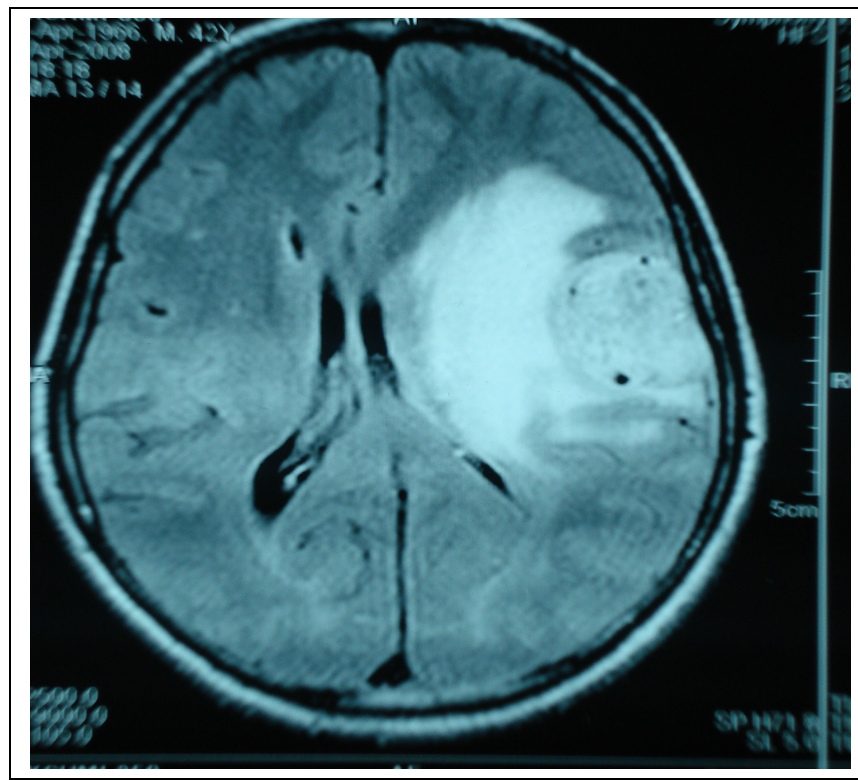
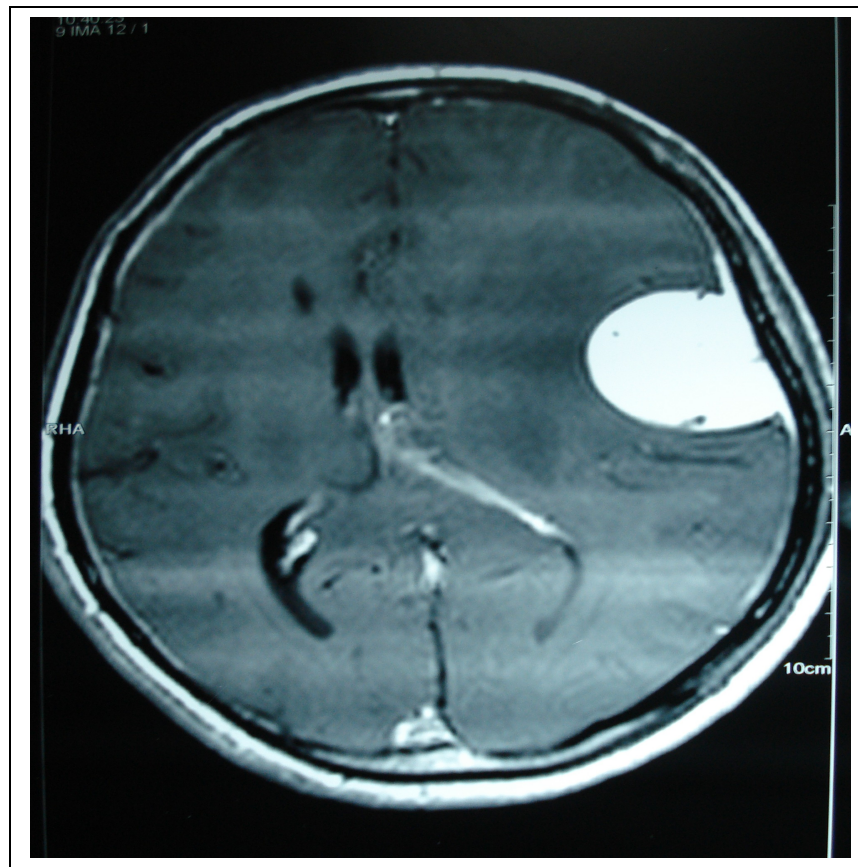


CHART - 9

ETIOLOGY PROFILES IN THE STUDY

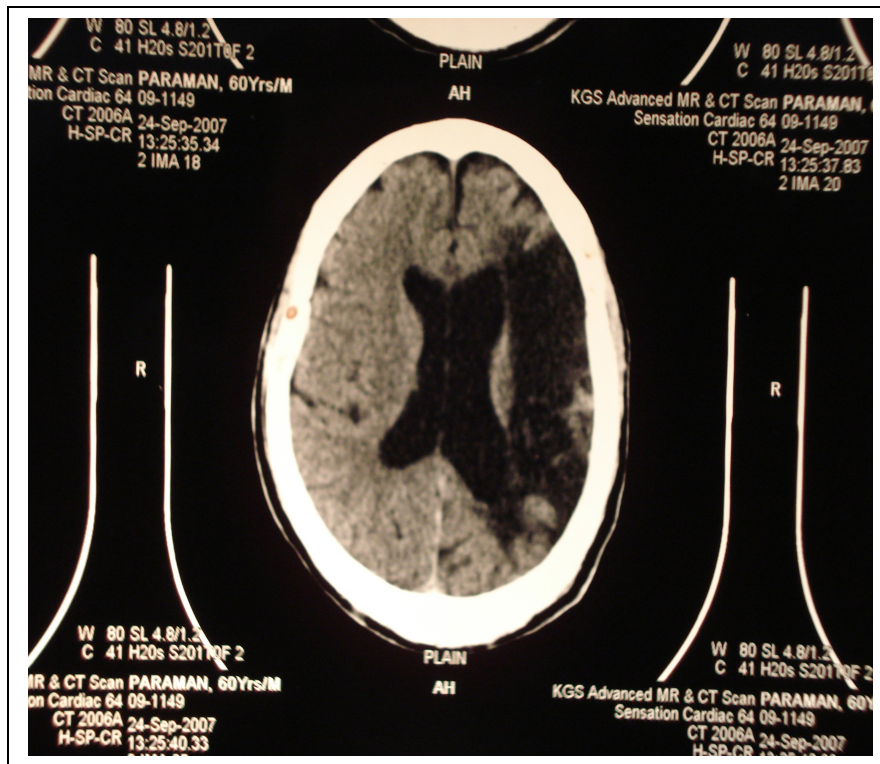


CASE OF MENINGIOMA

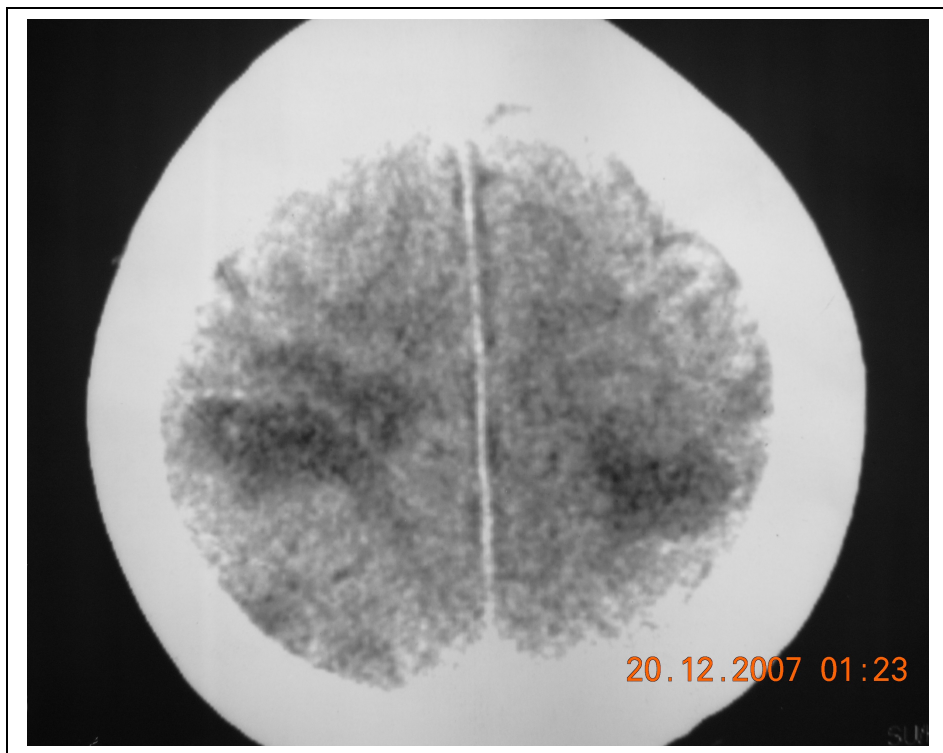


POST INFARCT SEIZURE

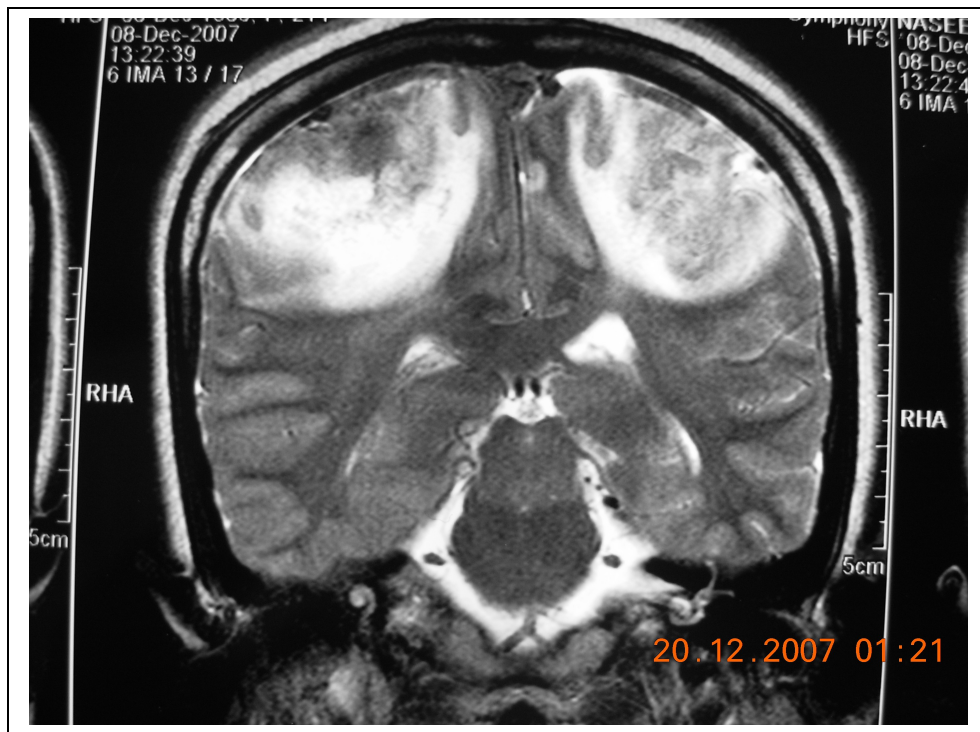




CEREBRAL VENOUS THROMBOSIS



CEREBRAL VENOUS THOROMBOSIS



REFERENCES

- 1) Epilepsy in the Elderly, Toshio Hiyoshi and Kazuichi Yagi, *Epilepsia* 41 (Suppl. 9) : 31-35, 2000.
- 2) Historical review of seizures and epilepsy, by E. Goldensohn, *Epilepsia*
- 3) *Neurology in Clinical Practice*, 4th Edition By Walter G. Bradley, DM, FRCP
- 4) *Harrison's principle of internal medicine* 16th ed.,
- 5) WHO fact sheet N °168 revised February 2001.
- 6) Hauser WA, Annegers JF, Kurland LT . Incidence of epilepsy and unprovoked seizures in Rochester, Minnesota; 1935 – 1984. *Epilepsia* 1993; 34:453- 68
- 7) foregren L, Bucht G, Eriksson S, Berg mark L. Incidence and clinical characterization of unprovoked seizures in adults: a prospective population-based study. *Epilepsia* 1996; 37: 224-29.
- 8) camfield PR, camfield CS ,Dooley JM ,Tibbles J, fung T, garner B. epilepsy after a first unprovoked childhood. *Neurology* 1985;35:1657-60.
- 9) Hauser WA, rich SS, lee JR annegers Jf ,Anderson VE. Risk of recurrent seizures after two unprovoked seizures. *N Engl J med* 1998;338:429-34.
- 10)shinnar S, berg AT, O'Dell newstein D, Moshe SL , hauser WA.predictors of

multiple , seizures in a cohort of children prospectively followed from the time of their first unprovoked seizures. Ann Neurol 2000;48:140-7.

11) ILAE Epilepsia 1989, 30, 389-399

12) VOL 111 / NO 1 / JANUARY 2002 / POSTGRADUATE MEDICINE

13) Kochiadakis GE, Rombola AT, Kanoupakis EM, et al. Assessment of autonomic function at rest and during tilt testing in patients with vasovagal syncope. Am Heart J 1997;134(3):459-66

14) Gates JR, Ramani V, Whalen S, et al. Ictal characteristics of pseudoseizures. Arch Neurol 1985;42(12):1183-

15) BMJ vol 332 11 feb 2006

16) Author: [William J Nowack, MD](#), Associate Professor, Department of Neurology, [University of Kansas Medical Center](#) e MEDICINE SPECIALITIES

17) Musicco M, Beghi E, Bordo B: Randomized clinical trial on the efficacy of antiepileptic drugs in reducing the risk of relapse after a first unprovoked tonic-clonic seizure. First Seizure Trial Group (FIR.S.T. Group). Neurology 1993 Mar; 43(3 Pt 1): 478-83

18) Annegers JF, Shirts SB, Hauser WA, Kurland LT: Risk of recurrence after an initial unprovoked seizure. Epilepsia 1986 Jan-Feb; 27(1): 43-50.

19) Hopkins A, Garman A, Clarke C: The first seizure in adult life. Value of clinical features, electroencephalography, and computerised tomographic scanning in prediction of seizure recurrence. Lancet 1988 Apr 2; 1(8588): 721-6

- 20) Hauser WA, Rich SS, Annegers JF, Anderson VE: Seizure recurrence after a 1st unprovoked seizure: an extended follow-up. *Neurology* 1990 Aug; 40(8): 1163-70.
- 21) Van Donselaar CA, Schimsheimer RJ, Geerts AT, Declerck AC: Value of the electroencephalogram in adult patients with untreated idiopathic first seizures. *Arch Neurol* 1992 Mar; 49(3): 231-7
- 22) Berg AT, Shinnar S: The risk of seizure recurrence following a first unprovoked seizure: a quantitative review. *Neurology* 1991 Jul; 41(7): 965-72
- 23) Beghi E, Berg AT, Hauser WA: Treatment of Single Seizures. In: Engel J Jr, Pedley TA, eds. *Epilepsy: A Comprehensive Textbook*. Philadelphia, Pa: Lippincott-Raven; 1997: 1287-1294.
- 24) Moore-Sledge CM: Evaluation and management of first seizures in adults. *Am Fam Physician* 1997 Sep 15; 56(4): 1113-20
- 25) Tardy B, Lafond P, Convers P, et al: Adult first generalized seizure: etiology, biological tests, EEG, CT scan, in an ED. *Am J Emerg Med* 1995 Jan; 13(1): 1-5
- 26) Bora I, Seckin B, Zarifoglu M, et al: Risk of recurrence after first unprovoked tonic-clonic seizure in adults. *J Neurol* 1995 Feb; 242(3): 157-63
- 27) Camfield PR, Camfield CS, Dooley JM, et al: Epilepsy after a first unprovoked seizure in childhood. *Neurology* 1985 Nov; 35(11): 1657-60
- 28) Chadwick D, Smith D: Epileptology of the first-seizure presentation. *Lancet* 1998 Dec 5; 352(9143): 1855; author reply 1856

- 29) Musicco M, Beghi E, Solari A, Viani F: Treatment of first tonic-clonic seizure does not improve the prognosis of epilepsy. First Seizure Trial Group (FIRST Group). *Neurology* 1997 Oct; 49(4): 991-8
- 30) King MA, Newton MR, Jackson GD, et al: Epileptology of the first-seizure presentation: a clinical, electroencephalographic, and magnetic resonance imaging study of 300 consecutive patients. *Lancet* 1998 Sep 26; 352
- 31) Walczak TS, Jayakar P. Interictal EEG. In: Engel J Jr, Pedley TA. *Epilepsy: a comprehensive textbook*. Philadelphia: Lippincott-Raven, 1997:831-48
- 32) Labovitz DL, Hauser WA, Sacco RL: Prevalence and predictors of early seizure and status epilepticus after first stroke. *Neurology* 2001 Jul 24; 57(2): 200-6
- 33) Zhu PG Neurology Department, Tong Ji Hospital, Tong Ji Medical university, Wuhan. PMID;2282883.
- 34) Perez lopez JL et al *Acta Neurol Scand*. 1985 Oct: 72 (4);380-4
- 35) De la Sayette V et al *Canadian Journal of neurosciences* Aug 1987;14(3);286-9.
- 36) Seizure disorders; the changes with age. *Epilepsia* 33 (suppl 4);S6-S14, 1992.

- 37) Chandra B: First seizure in adults: to treat or not to treat. Clin Neurol Neurosurg 1992; 94 Suppl: S61-3
- 38) Davidson DL: What to do with the first fits. Scott Med J 1999 Feb; 44(1): 6-8.
- 39) Fisher RS: Imitators of Epilepsy. New York, NY: Demos Publications; 1994: 372.
- 40) Hart YM, Sander JW, Johnson AL, Shorvon SD: National General Practice Study of Epilepsy: recurrence after a first seizure. Lancet 1990 Nov 24; 336(8726): 1271-4
- 41) Heller AJ, Chesterman P, Elwes RD, et al: Phenobarbitone, phenytoin, carbamazepine, or sodium valproate for newly diagnosed adult epilepsy: a randomised comparative monotherapy trial. J Neurol Neurosurg Psychiatry 1995 Jan; 58(1): 44-50
- 42) Jallon P, Hauser A, Roman GC: Guidelines for epidemiologic studies on epilepsy. Epilepsia 1993; 34(4): 592-596 Johnson LC, DeBolt WL, Long MT, et al: Diagnostic factors in adult males following initial seizures. A three-year follow-up. Arch Neurol 1972 Sep; 27(3): 193-7
- 43) Kotsopoulos IA, de Krom MC, Kessels FG, et al: The diagnosis of epileptic and

non-epileptic seizures. *Epilepsy Res* 2003 Nov; 57(1): 59-67

44) Leppik IE: Contemporary Diagnosis and Management of the Patient with Epilepsy, Fifth Edition. Newtown, Pa: Handbooks in Health Care; 2000.

45) Liporace JD, Sperling MR: Simple autonomic seizures. In: Engel J Jr, Pedley TA, eds. *Epilepsy: A Comprehensive Textbook*. Philadelphia, Pa: Lippincott-Raven; 1997: 549-555.

46) Martinovic Z, Jovic N: Seizure recurrence after a first generalized tonic-clonic seizure, in children, adolescents and young adults. *Seizure* 1997 Dec; 6(6): 461-5.

47) Mattson RH, Cramer JA, Collins JF, et al: Comparison of carbamazepine, phenobarbital, phenytoin, and primidone in partial and secondarily generalized tonic-clonic seizures. *N Engl J Med* 1985 Jul 18; 313(3): 145-51.

PROFORMA FOR EVALUATION

Name

Age

Sex

Address

Occupation

Contact no

Medical unit prof

op/ip no

ward

DOA

DOD

Neuro unit prof

min no

EEG no

Referred from

Elaboration of seizure

Witnesse of seizure

Reliability

Before the episode

Recent illness (headache / fever)

Unusual stress

Medications

Last alcohol intake

Last meal

Sleep deprivation

Activity just before seizure

During the episode

Time of day

Aura

Duration

Ability to talk & comprehend

Ability to recall events

Movements of eyes face arms legs

Tongue bite frothing

Bowel / bladder incontinence

Bodily injuries sustained

After event

Confusion duration

Focal neurological deficits

Any other significant symptoms

Diabetic : yes / no duration & treatment

CKD

any others

smoking

Clinical Examination

Vitals :	BP	Pulse	RR	Temp
----------	----	-------	----	------

P/A :

COURSE DURING HOSPITAL STAY

INVESTIGATIONS :

Hematology **TC :** **DC:** **P L E B** **HB :** **ESR:**

Biochemistry **sugar** **urea** **creatinine** **Na** **k** **Ca**

CSF analysis

Others :

ECG :

Cxr :

ECHO :

CT BRAIN :

MRI BRAIN :

EEG :

Treatment

ABBREVIATIONS AND ACRONYMS

EEG	-	Electro EncephaloGram.
CSF	-	Cerebro Spinal Fluid.
CT	-	Computerized Tomogram.
MRI	-	Magnetic Resonance Imaging.
ILAE	-	International League Against Epilepsy
GTCS	-	Generalized Tonic-Clonic Seizure
CVT	-	Cortical Venous Thrombosis
CVA	-	Cerebro Vascular Accidents.
CP angle	-	Cerebello Pontine Angle.
AED	-	Anti Epileptic Drug.
SDH	-	Sub Dural Hemorrhage.

MASTER CHART

S.No.	Name	Age	Sex	SZT	HO R	PPF	HA D	NE A	ME T	CTS	EEG
Cerebro Vascular Accident											
1	Irudaya Raja	52	M	G	Y	Y	U	Ab	N	A	A
2	Paraman	60	M	P	Y	Y	S	Ab	N	A	A
3	Saradha	51	F	P	Y	N	S	Ab	A	N	A
4	Ameer Sultan	59	M	G	Y	N	U	Ab	A	A	A
5	Fakir Ahmed	51	M	P	Y	N	S	Ab	A	A	A
6	Vellaisamy	63	M	G	Y	Y	S	Ab	N	A	A
7	Meenakshi	59	F	G	Y	N	S	Ab	A	A	A
8	Rajathee	58	F	G	Y	N	U	Ab	N	A	A
9	Pitchai	53	M	G	Y	Y	S	Ab	A	A	A
10	Ibragim	67	M	P	Y	N	S	Ab	N	A	A
11	Muniyandi	57	M	G	Y	N	S	Ab	N	N	A
12	Damodaran	64	M	P	N	N	S	Ab	A	A	A
13	Laola Thevar	54	M	P	N	Y	S	Ab	A	A	A
14	Bhanumathi	52	F	G	Y	N	S	Ab	A	A	A
15	Akbar Hussain	68	M	G	Y	N	S	Ab	A	A	A
16	Durai raj	70	M	G	Y	N	S	Ab	A	A	A
17	Vellaisamy	69	M	G	Y	N	S	Ab	A	A	A
18	Chandra	50	F	G	Y	N	S	Ab	A	A	N
19	Tamilarasi	58	F	G	Y	N	U	Ab	N	A	A
20	Sekar	57	M	P	Y	N	U	Ab	A	A	A
21	Alagammal	56	F	G	Y	N	U	Ab	A	A	-
22	Vatsala	57	F	P	Y	N	S	Ab	N	N	-
TUMOR											
23	Maruthiah	69	M	G	Y	Y	S	N	A	A	A
24	Parvathy	57	F	G	Y	N	S	N	N	A	A
25	Ganesan	53	M	P	Y	N	S	N	N	A	A

64	Arumugam	55	M	G	Y	N	S	Ab	N	N	A
65	Jeya laxmi	51	F	G	Y	Y	U	Ab	A	N	A
66	Chellammal	51	F	P	Y	Y	S	Ab	A	N	N
CVT											
67	Rengaraj	59	M	P	Y	Y	U	Ab	N	N	-
68	Navaneethan	67	M	G	Y	N	S	Ab	N	N	A
Idiopathic											
69	Karuppasamy	69	M	G	N	Y	S	Ab	N	N	A
70	Thangam	53	F	G	Y	Y	U	N	A	N	N
71	Veluchamy	64	M	P	Y	N	U	Ab	N	N	N
72	Raja	60	M	G	N	Y	S	N	A	A	N
73	Gandhimathi	72	F	G	N	N	S	N	A	N	A
74	Kanagaraj	51	M	P	N	N	S	N	N	A	A
75	Muniyandi	65	M	G	Y	Y	S	N	A	N	N
76	Vasantha	52	F	G	Y	N	U	N	N	A	N
77	Pandiammal	61	F	G	Y	Y	S	N	A	N	A
78	Subbiah	63	M	G	Y	N	S	N	A	A	A
79	Vazhavanthal	63	F	G	N	Y	S	N	N	N	N
80	Pitchaiammal	61	F	G	Y	N	S	N	A	A	A
Sub Dural Hematoma											
81	Rengachari	72	M	P	Y	N	U	Ab	A	N	A
82	Durai raj	74	M	G	Y	N	S	Ab	N	A	A

KEY TO MASTER CHART

SZT - SEIZURE TYPE

HOR	-	HISTORY SUGGESTIVE OF ORGANICITY
HAD	-	HOMODYNAMIC STATUS AT ADMISSION
NEA	-	NEUROLOGICAL EXAMINATION AT ADMISSION
MEAT	-	METABOLIC ABNORMALITIES AT ADMISSION
CTS	-	CT SCAN
M	-	MALE
F	-	FEMALE
P	-	PARTIAL SEIZURE
Y	-	YES
N	-	NO
S	-	STABLE
U	-	UNSTABLE
N	-	NORMAL
A	-	ABNORMAL